Campylobacteriosis rates show age-related static bimodal and seasonality trends

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Abstract

Aim Campylobacteriosis is highly characterised by a strongly seasonal rate of incidence. Age is also known to be a risk factor for sporadic campylobacteriosis, but little has been done to quantify age-related rates of campylobacteriosis. This study investigates age-related incidence across countries and up to 12 years of data, as well as differences in seasonality within age groups.

Methods Graphical and statistical analysis of officially collected campylobacteriosis reports from three countries available from official websites.

Results For Australia, New Zealand and Canada, rates of campylobacteriosis show marked peaks at <4 years and 20–29 year age bands. These peaks indicate that stable age-related factors impact on campylobacteriosis epidemiology in all three countries. Seasonality is expressed differently across these age bands, and in years of extremes of incidence.

Conclusion Campylobacteriosis is highly seasonal, but overlying this is a stable age-related pattern of incidence, with two peaks approximately 20 years apart. Highest seasonal differences occur with ages between the two peaks.

Campylobacteriosis is highly prevalent in New Zealand, with rates significantly above rates in other temperate countries. Notwithstanding the recent marked drop in campylobacteriosis reported in New Zealand coinciding with implementation of a chicken health scheme, other trends in campylobacteriosis rates indicate factors beyond chicken meat at play.

A strong seasonal pattern, typically an early winter low and an early summer high rate of incidence, is common to New Zealand and other temperate countries. At an annual level, there is a clear correlation between increased consumption of chicken meat and the rates of reported campylobacteriosis, seemingly confirming that the route to humans is via consumption of contaminated chicken, particularly as the incidence of Campylobacter in chickens has a similar seasonal pattern. However, this correlation disappears on considering shorter time intervals.

Specifically, chicken consumption is not seasonal and the seasonal peak in chicken contamination typically occurs after the human peak, leading us to suggest a domestic fly-related epidemiology via fomites and fingers to food. Difficulty with the chicken/human link was also found in a five year German study where the human seasonal incidence preceded that of chickens.

Regional and seasonal variations in campylobacteriosis have been reported through New Zealand, particularly differing seasonal incidence patterns with changing latitude. Similar differences have been reported across Northern Europe.
Age-related trends have also been noted, and an acquired immunity has been suggested as a reason for the rapid drop-off in rates of infection with age, supported by observation that children older than six months tend not to develop diarrhoea except on first exposure. Seasonal peaks and higher incidence in children under five years has also been noted in the United Kingdom.

In a rural/urban comparison in Canada, differences in rates of infection were noted not only for babies, but again at adult-onset, although no exploration of this observation was made. A similar twin age-related peak in rates in New Zealand has been noted and this investigation aims to determine the reality and stability of this observation, and to attempt to elucidate an epidemiological profile to account for it.

**Methods**

Epidemiological data were obtained primarily from on-line resources, www.nzpho.org.nz for New Zealand, http://www9.health.gov.au for Australia and http://dsol-smcd.hc-sc.gc.ca for Canada, accessed November 2009. In all three countries, campylobacteriosis rates (case numbers per 100,000 population) are provided. Data is offered in different formats and broken into various categories.

Data was analysed to determine trends in rates over years using rates of reported illness from the on-line resources that are expected to have been normalised for population changes during the reporting period. The official data reports use different demographic fractions, especially the age bands. These have been normalised where comparisons required it, but otherwise are reported in their native format. Years of data availability also vary across countries. We used the full 12-year period of 1997–2008 for New Zealand, 6 years, 2003–2008 for Australia, and 7 years 1998–2004 for Canada. Data is graphed to show mean, high and low values for the age bands and year periods covered.

Further data was requested from the New Zealand Public Health Observatory’s EpiSurv database in more detail than that available through their on-line query tool. Cases by age class by month data were obtained for the years 1997-2008. The Gini coefficient, a measure of inequality of distribution, by age band average over the 12 years of data was used as a measure of seasonality.

Privacy and ethics. No data source offers information identifiable to an individual.

**Results**

Reported campylobacteriosis rates are age-dependent and can vary quite markedly across the age bands. As noted in previous studies, very young children typically exhibit high rates of campylobacteriosis, and this is clearly evident in Figure 1 across all three countries.

A Canadian study indicated, but did not explore further, a second peak at adult-onset. This is also clearly evident in Figure 1 across all three countries. Furthermore, these peaks in reported rates are approximately 200% and 150% for 0–4 years and 20–29 years bands respectively above the background rates for other age bands in each country, even though those background rates are markedly dissimilar.

Australian rates are less than half those of New Zealand, and Canadian rates a third lower again. Clearly the presence of two peaks in campylobacteriosis rates of infection approximately 20 years apart is not an artifact of the very high New Zealand incidence. Even picking the highest and lowest rate for each age group across the years represented does not change the marked presence of these two age-related peaks.

The stability of these age-related peaks in incidence across many years of data and three countries strongly indicates a difference in epidemiology is occurring. Figure 2 shows detail of the stability of these peaks for New Zealand. Note that this graph
covers a 12-year period, clearly indicating an age-related impact rather than a specific group of people more or less susceptible to campylobacteriosis.

Figure 1. Campylobacteriosis rates (cases per 100,000 population) at different age bands for countries as marked. New Zealand n=12 years 1997–2008; Australia n=6 years 2003–2008; Canada n=7 years 1998–2004

Note: High is the highest rate over the period and low the lowest, regardless of year.
Figure 2. Campylobacteriosis rates (cases per 100,000) in New Zealand at age bands per year reported for the years 1997 to 2008

On a population basis, rates increase in the 1–4 year age band over the <1 year band (data not shown in Figure 1 as these have been amalgamated, but are available separately for Canada and New Zealand). Interestingly, the seasonality impact between these two age groups is markedly different, with the <1 age group showing the least seasonal impact as indicated by the low Gini coefficient in Figure 3. This group is presumably the most susceptible to any Campylobacter exposure and also most likely to be reported.

Although the 5–9 year age group has a low incidence, they typically exhibit the highest seasonality. Of interest too is that the pattern of seasonal impact in New Zealand changes dramatically for the 2008 year, and that the highest and lowest rates of incidence years in this series (2006 and 2008) exhibit the lowest and highest seasonal differences respectively across all age groups.
Figure 3. Gini coefficient for monthly New Zealand data averaged over the years 1997–2008, and for the years 2006 (highest incidence year) and 2008 (lowest incidence year) separately

Note: This coefficient approaches zero as seasonality decreases. It is a measure of the inequality of a distribution, a value of 0 expressing total equality and a value of 1 maximal inequality. It can be multiplied by 100 to range between 0 and 100.

Discussion

These data do not appear to indicate the presence of any ongoing acquired immunity with age as may be expected after an early childhood primary infection because of this clearly age-specific static 2nd peak, although short-term immunity can play a role in campylobacteriosis disease expression.8,11,16

As Campylobacter is relatively ubiquitous in the environment (in animals, wild birds, water and food including poultry), humans can expect relatively frequent ongoing exposures to it regardless of age. This would then be expected to provide an ongoing immunity booster effect to any earlier childhood exposure infections and provide an ongoing intermittently boosted acquired immunity – but it does not for the 20–29 year age group only. It seems likely there must be another explanation rather than loss of acquired immunity. Similarly the absence of a third peak another 20 years later, or shorter for compromised immunity with age, further detracts from the likelihood of a loss of acquired immunity explanation.

Acquired immunity is interesting, although the antibody literature is hard to interpret because of differing methods.8 Antibodies appear to drop off over a 12-month period leading to the observation that the presence of antibodies indicates a Campylobacter
challenge in the preceding year. If this is a general population exposure, it is hard to suggest a reason for the marked increase in reporting rates in early adulthood, and how an immunity reaction could impact on changing seasonality expression for different age bands. Why would frequent exposure, suggested by the antibody studies, result in different disease expression rates for different age bands?

An epidemiological explanation for the lower rates of <1 year and higher rates in the 1-4 age group is easily made. Babies are likely to have a low exposure level, but this rises as they reach teething and become mobile. The subsequent reduction in rates for higher age groups could be explained by an acquired immunity reaction if it was not for this second peak in rates only in the early adult years.

If chicken-consumption is truly the source for most cases, why did rates generally decrease in 2007, before the chicken health scheme began, and what could be the link between chicken-consumption and age, regardless of sex? Further, it is difficult to reconcile chicken consumption with the marked and long-standing reduced seasonality of the 5-9 year band (high Gini coefficient) compared to the other age groups.

The bimodal peaks in rates demonstrated here combined with the age-related differences in seasonality of cases is not readily explained by exposure to chicken products nor to current immunological observations. It is possible that the second 20-29 year old peak reflects high rates of primary infection across this age band, but again it is difficult to provide an explanation that would increase exposure to just them and not any other age band, for instance teenagers.

The 2006 year coincides with an unusually high New Zealand rate of cases in autumn/winter, subsequently found to be associated with a rare sequence type.17

**Conclusion**

The epidemiology of campylobacteriosis is complex. The presence of distinct differences in rates of infection for age groups that remain stable over long periods of time indicate a substantial underlying factor. Further, seasonal factors clearly play different roles at different ages, and the markedly different seasonality expressed in the New Zealand peak year (2006) and lowest year (2008) suggest scope for further investigation. Until specific exposure and susceptibility explanations can be made to account for these age-related differences, the popular assumption that poultry is the primary source for human campylobacteriosis is perhaps rather simplistic.

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